All muck and no brass

Evidence-based diagnostic testing is such barren ground. Most studies of diagnostic tests are so flawed that systematic reviews of diagnostic tests are bonkers: they just measure how high the muck is piled. They can't tell us whether there's brass in the muck.

This month's *Bandolier* concentrates on diagnostic testing. It may not be obvious, but there is a theme about clinical signs and symptoms and clinical scoring systems. The theme also extends to using information from clinical trials to generate useful diagnostic criteria. This extends ideas extolled in the Internet version of *Bandolier* in a web essay on diagnostics and it is delightful to see good examples.

Predicting risk of cardiovascular disease and a diagnostic tool for erectile function in primary care both come directly from randomised trial data. Diagnostic decision rules are not often tested, but there is a comparative test to help decide which rule to use for sending women for bone density measurements. And there's information about not being flummoxed by trigger points in fibromyalgia.

Palliative care

The *Bandolier* Palliative care Internet site this month examines meta-analyses of treatments for chemotherapy and radiotherapy-induced nausea and vomiting. Several things of note. First that there are four good meta-analyses in this arcane area, and second that when put together it all makes sense. Some treatments are better than others, and choices can be facilitated by a quick examination of all relevant evidence in one place.

Electronic Bandolier

Monthly visitors to the site are now just under 400,000. They come from all over the world, even if it's only one or two from Andorra, Uganda and Nepal. What's fascinating is the incredible spread of stories visited. There's more this month on healthy living and headache and migraine. By the Autumn we hope to be able to announce an even bigger electronic effort. If you want a monthly email about what's new at the *Bandolier* Internet site, just drop an electron to bandolier@pru.ox.ac.uk.

You can't dam half a stream

Interesting stuff from Boston Consulting Group on whether interventions to inhibit prescribing ever work. Many references to read, but maybe another area to explore.

FIBROMYALGIA:

DIAGNOSIS AND TREATMENT

Readers who dart to this article in the hope that someone has cracked the problem are going to be disappointed. Those fortified with some Scottish medicine may have the strength to read a little about diagnostic difficulty and therapeutic back-up.

Background

Fibromyalgia, for *Bandolier*, has been a bit like calcium metabolism. You know it's there. You know that it's important, but time is somehow never quite right to turn one's brain on and get stuck in. On the whole, we'd rather be in Philadelphia.

The trouble is that there's a lot of fibromyalgia around. About 10% of the population may have chronic widespread pain, with the more serious diagnosis of fibromyalgia in 1-2% or so, but more in people over 60. That's getting on for 10,000 and 2,000 patients respectively for an average Primary Care Group of 100,000 people. Diagnosis is difficult and treatments limited.

Diagnosing fibromyalgia

The American College of Rheumatology has produced diagnostic guidelines. The historical feature of fibromyalgia is widespread pain of three months duration or more affecting the axial skeleton and at least two contralateral quadrants of the body. In addition, for a diagnosis of fibromyalgia patients have to feel pain in 11 of 18 trigger points when they are palpated with the amount of pressure sufficient to blanch a finger nail.

How useful are trigger points? Tricky territory this, because large data sets seem to be lacking. In Cheshire, 250 subjects

*The views expressed in **Bandolier** are those of the authors, and are not necessarily those of the NHSE*

Bandolier's **electronic resources at www.ebandolier.com**

selected from participants in a chronic pain survey were asked to undergo a trigger point examination [1]. Of the 250, 100 had chronic widespread pain, 100 regional pain and 50 no chronic pain in the survey. On the day of examination, pain state in these patients had shifted. For instance 3/74 with stated chronic widespread pain now had no pain, and 7/39 with no chronic pain now had chronic widespread pain.

According to their status on the day of examination, the criterion of 11 of 18 painful trigger points was found in 40% of patients with chronic widespread pain, 20% of those with regional pain and 5% of those with no pain (Figure 1). Overall 20 of 132 patients without one of the two criteria of fibromyalgia had at least 11 painful trigger points. By contrast 29% of patients with chronic widespread pain had only 0-4 painful points.

The criterion of eleven painful trigger points looks a poor diagnostic bet. The problem with assessing painful trigger points are several. There is no gold standard against which they can be measured. Experts elicit different numbers [2] in the same patients. Again, perhaps an all or nothing approach to fibromyalgia (with or without trigger points) hides a wider spectrum of disease.

Antidepressants for fibromyalgia

Readers should be looking forward to a feast when they hear that three meta-analyses have been published in recent years [3-5]. Unfortunately these only serve up a light snack. Partly that is to do with the raw materials, in that trials have been often small (most with 6-47 patients), some have had poor diagnostic criteria, and many report outcomes in ways that are unhelpful. Partly, though, it's to do with the way that the raw materials have been combined. Only one review [3] tells us about the reporting quality and validity of the trials being combined, making the others somewhat less than useful except as a source of references.

What can be said is that antidepressants have been used for treating fibromyalgia successfully [3]. Quality in 13 included trials was good, initial diagnosis predominantly used American College of Rheumatology criteria or its equivalent, and that outcomes were sensibly reported in most.

Results

To improve the symptoms of one patient with fibromyalgia, we have to treat four (95%CI 2.9 to 6.3) with antidepressants rather than placebo. Improvements for fatigue, sleep, overall well-being and pain were significant and moderate using effect sizes. Painful trigger points were not improved.

Comment

There's no neat answer here, only more questions. We use the criterion of 11 painful trigger points to define fibromyalgia, but the most successful treatment does not change the number of trigger points. It helps pain, sleep and other symptoms, and maybe patient and physician assessment of problems, but not the diagnostically defining criterion.

This is food for thought, that meta-analysis of treatment studies can help the understanding of diagnosis. The evidence for the usefulness of trigger points is thin. The evidence that antidepressants help is moderate. But we still don't know what dose of what drug is best for whom, or what we are treating in a disorder that may affect a lot of us. Great when things come together, isn't it?

References:
1   P Croft et al. Population study of tender points counts and pain as evidence of fibromyalgia. BMJ 1994 309: 696-699.
2   F Wolfe et al. The fibromyalgia and myofascial pain syndromes: a preliminary study of tender points and trigger points in persons with fibromyalgia, myofascial pain syndrome and no disease. Journal of Rheumatology 1992 19: 944-951.
3   PG O'Malley et al. Treatment of fibromyalgia with antidepressants: a meta-analysis. Journal of General Internal Medicine 2000 15: 659-666.
4   LM Arnold et al. Antidepressant treatment of fibromyalgia: a meta-analysis and review. Psychosomatics 2000 41: 104-113.
5   LA Rossy et al. A meta-analysis of fibromyalgia treatment interventions. Annals of Behavioural Medicine 1999 21: 180-191.

Figure 1: Trigger points in chronic widespread pain, regional pain and no pain
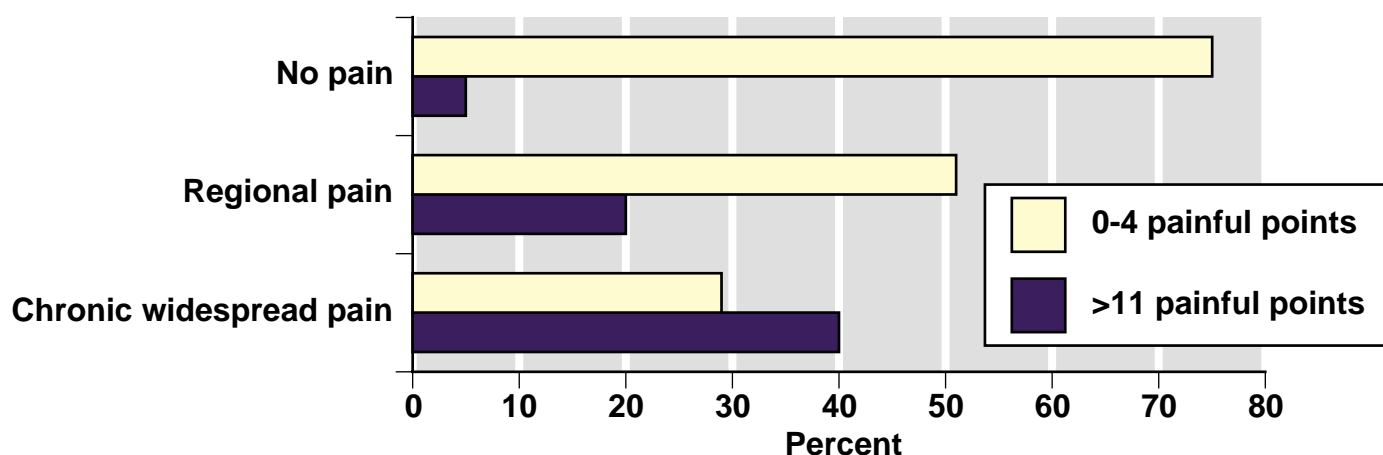
ACUPUNCTURE FOR FIBROMYALGIA

Because fibromyalgia is a difficult painful condition with no easy treatment options, acupuncture is often used. One reason is that "patients want it". One wonders whether patients are fully informed. So what is the evidence that acupuncture is effective? A review tells that there is little or no evidence of benefit, but that it might make things worse [1].

Review

The review was done by a complementary medicine group in Baltimore with a deserved reputation for good work. In particular, searching was exemplary, using not only electronic database searching, but special registries, their own registry of complementary medicine in pain and letters to nearly 100 specialised institutions or individuals.

Results

There were three randomised trials, three prospective cohort studies and one retrospective cohort study. The three randomised trials involved 135 patients. Two were of low methodological quality. All the cohort studies were deemed to be of low quality. So that left one randomised trial (done in Geneva [2]) that compared electro-acupuncture with sham electro-acupuncture over three weeks in 70 patients, with no long term outcomes. Acupuncture was reported to be better than sham acupuncture for several outcomes, but there were differences in sex split and duration of disease at baseline, so that it looked like a randomisation failure.

Comment

Does this amount to a row of beans? The review implies that it does, and talks about implications for practice.

Would this level of evidence be acceptable for a new pharmacological or surgical procedure? It would not. The double standards seen from proponents of complementary medicine is really worrying. Unproven treatments are being used that can be harmful, and the review comments that some patients suffered from exacerbation of symptoms.

The use of evidence should be qualitative first and quantitative second. That is why we spend so much time and effort looking for sources of bias. We are rightly concerned about any bias from pharmaceutical companies selling their wares. Should we not equally be concerned when reviews from special interest groups accept any old evidence and ignore the limits we impose for other treatments? And who tells patients that acupuncture for fibromyalgia has limited evidence for effectiveness, but can do real harm? And if not, why not? It should be a lawyer's paradise.

References:
1   BM Berman et al. Is acupuncture effective in the treatment of fibromyalgia? Journal of Family Practice 1999 48:213-218.
2   C Deluze et al. Electroacupuncture in fibromyalgia: results of a controlled trial. BMJ 1992 305:1249-1252.

DEXAMETHASONE AND CHEMOTHERAPY NAUSEA AND VOMITING

From time to time along comes a paper that makes you wish that you had written it. That might be because it will make you famous (but who wants reporters nagging them anyway?). It's more likely to be because it was a smashing bit of work that was helpful and well done. One such is an examination of dexamethasone used to control chemotherapy-induced nausea and vomiting [1].

This may not be a topic that affects many as professionals, but for patients it is important. It is also important because palliative care has too few good trials, let alone good systematic review, so exemplars matter.

Background

Start by thinking of the problems that a review might encounter. First is the setting. Different chemotherapy regimens are thought to have different effects on emesis, with some being highly emetogenic and others less so. The patients may be having a first cycle, or have had some cycles before, and may or may not have had previous chemotherapy. They may have a history of chemotherapy-induced emesis. The type of cancer may be the same or different. The age of the patients may differ.

Then there is the treatment and the comparator. Dexamethasone dose may vary, as well as the mode of delivery. The comparator may be placebo, but that would be unfair, so it may be placebo with cover from other antiemetic, or other antiemetic, or combinations.

Then there is the period over which emesis is measured. Is the first 24 hours different from later periods, and over what period is it important to measure any effect of dexamethasone?

Then there is what you measure. Is it vomiting alone, or nausea alone, or retching, or complete control of emesis?

Then there is study design and reporting. Do the trials have properties that may confer bias? Are the studies using valid methods?

Anyone undertaking such a review might be forgiven for feeling a little nauseated at the prospect of sorting this lot out. Is it possible to make sense of it all?

Review

The review sought randomised trials of dexamethasone for controlling chemotherapy-induced nausea and vomiting using a variety of electronic databases, as well as hand searching journals. Comparison was with placebo, no treatment, active agents or combinations of these, and a number of different languages was permitted.

The main outcome was prevention of vomiting because that is more objective than nausea, but prevention of nausea was

also considered. Periods were split between the first 24 hours (acute phase) and up to 8 days after chemotherapy (delayed phase).

Results

There were 32 studies included with 42 different comparisons and information on 5,500 patients. Doses of dexamethasone varied between 8 mg and 100 mg. Half used 20 mg for the first 24 hours, and the mean dose over the acute and delayed phase was 56 mg. Information was collected about type of patient, cancer, chemotherapy, previous cycles, prior chemotherapy and history of chemotherapy-induced emesis. Comparison was predominantly with placebo or no treatment supplemented with other antiemetic agents.

The overall results for control of emesis in the acute phase are shown in Figure 1. With dexamethasone 1320/1911 patients (69%) did not vomit (and 31% did vomit) compared with 937/1713 patients (55%) who did not vomit (and 45% who did vomit) with control. The relative benefit was 1.3 (1.2 to 1.4) and NNT 7 (6 to 9).

The overall results for control of emesis in the delayed phase are shown in Figure 2. With dexamethasone 887/1461 patients (61%) did not vomit compared with 754/1679 patients (45%) with control. The relative benefit was 1.3 (1.2 to 1.5) and NNT 6 (5 to 8).

Comment

What was interesting was that both random effects and fixed effects were used in calculating odds ratios, risk ratios and risk difference, with a number of comparisons and end points. Potential variability in antiemetic efficacy was examined by looking at doses. Meta-regression, a complex statistical approach for most of us, was used to examine potential effects of trial design (blinding), and concealment of treatment allocation, and of different settings like moderately and highly emetogenic treatments. None made much difference. To some extent the consistency of response can be seen in the L'Abbé plots (Figures 1 and 2), both for big studies (largee symbols) and small studies (smaller circles).

The sensitivity analysis underpinned the overall conclusion that about six patients need to be treated to prevent one from experiencing emesis in either phase.

The value of the meta-analysis is not just in demonstrating that dexamethasone treatment works, and how well it works, but in the superb methodological workup to give us confidence in that result. An excellent example of a paper that could be used for training in what makes for acceptable standards in preparing and writing systematic reviews.

A wider perspective

On its Internet site, *Bandolier* also examines a series of other systematic reviews concerned with treatment of acute vomiting after chemotherapy and radiotherapy. High dose metoclopramide and conventional treatment do relatively

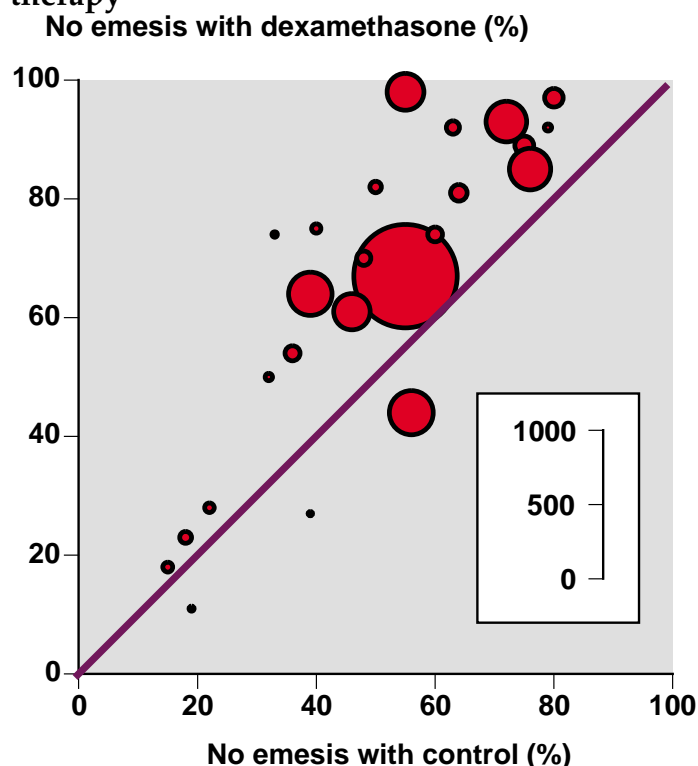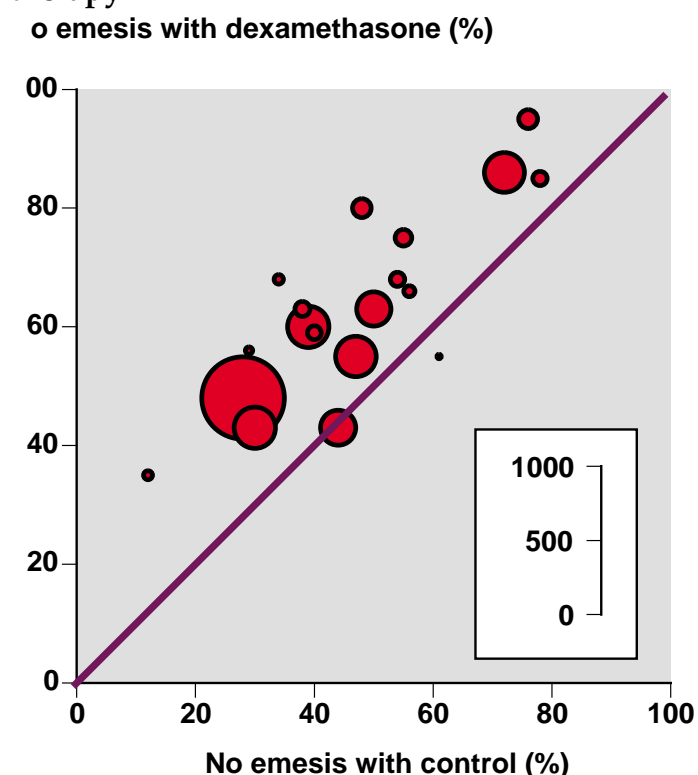**Figure 1: Acute phase: first 24 hours of chemotherapy**

Figure 2: Delayed phase: 2-5 days after chemotherapy

poorly, with about 50% vomiting. With 5HT-3 receptor antagonists alone about 40% vomit, while with 5HT-3 receptor antagonists plus dexamethasone, only 25% vomit. A nice example of relative efficacy in a difficult area.

References:
1   JP Ioannidis et al. Contribution of dexamethasone to control of chemotherapy-induced nausea and vomiting: a meta-analysis of randomised evidence. Journal of Clinical Oncology 2000 18: 3409-3422.

PREDICTION FROM RCTs

Bandolier has long wondered why information from randomised trials is not better used (or even used at all) to inform us about diagnosis or prognosis. When talking to professionals, they will often comment that they want less information about what treatment to use, but much, much more information about which patient to use the treatment on. Diagnosis, or risk assessment, is the key, especially in busy primary care.

The curious thing is that the answer to their prayers probably lies in the very randomised trials that tell them about treatment. Think about it for a moment. Trials of treatment A in disease X have to recruit patients with disease X. Many are screened, and some chosen. So first of all we have a mass of information about those who "have" the disease according to some contemporary definition, those who "do not have" the disease, and those who, treated or untreated, have desirable or undesirable outcomes. Despite the complexity of any diagnosis, surely someone could analyse this mass of information and tell us whether there is some simple rule that could guide those of us not involved in clinical trials to decisions about who to treat?

Alas not, or at least only too rarely. The reasons are about as complex as the trials and statistics, but it comes down to the fact that in healthcare companies the Cerebrus-like creatures who guard the data can't see why diagnosis is so difficult. That's what it's like in industrial circles, but other areas are more open, and can show the rest how to do it [1].

Study

The study [1] was based on an individual patient data analysis of antihypertensive intervention trials. This includes all major randomised trials of antihypertensive drugs for which individual patient information was available in 1995. There were 47,000 individuals. Of these, 3,001 had died in the average follow up of 5.2 years, and 1639 had died from cardiovascular causes.

In the eight trials, 16 baseline factors were common, and with prior plausibility as risk factors. These were:

- ♦ Age
- ♦ Sex
- ♦ Height
- ♦ Body mass index
- ♦ Current cigarette smoking
- ♦ Systolic and diastolic blood pressure
- ♦ Heart rate
- ♦ Total serum cholesterol
- ♦ Serum creatinine
- ♦ Serum uric acid
- ♦ Previous myocardial infarction
- ♦ Previous stroke
- ♦ Diabetes
- ♦ Left ventricular hypertrophy
- ♦ Treatment

Figure 1: Cardiovascular mortality and risk score
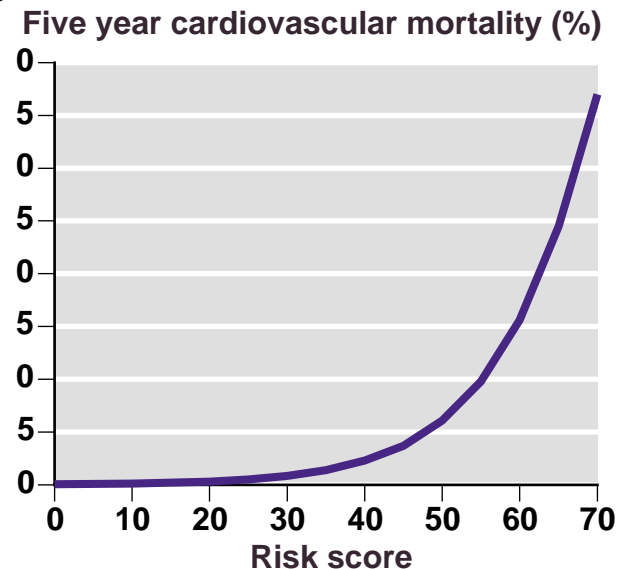
Multivariate analysis showed that BMI, diastolic blood pressure, heart rate and uric acid were non-significant predictors. The final model used the 12 remaining factors, and these were grouped into convenient intervals of blood pressure or cholesterol.

Results

The relationship between the total risk score from adding all the individual factors, and the risk of cardiovascular death over five years, is shown in Figure 1. For instance, a man (12 points) aged 54 (11 points) who did not smoke cigarettes (0 points), was 1.7 metres tall (3 points) with a systolic blood pressure of 130 (2 points), total cholesterol of 5.4 mmol/L (2 points) and creatinine of 80 mmol/L (1 point), and with no history of heart disease, stroke or diabetes, would have a total score of 31 points. That would translate into a five year risk of cardiovascular death of about 1%.

The scoring system is well laid out in the original BMJ article and its Internet site. Best of all is that there is an Internet site (www.riskscore.org.uk) available for users. It is easy to input information and it gives instant calculation of score and risk for an individual and the normal for age and sex.

Comment

The results are interesting, and most readers would want to know their own personal risk. If high, especially compared with normal for age and sex, something may be done about some of the risk factors, and the balance of risk reduction and inconvenience of doing anything weighed.

More important is the fact that this work has been done. It is an important exemplar of what professionals want, and how information can be used. Holders of information databases might want to take note, and get a dose of vision.

References:
1   SJ Pocock. A score for predicting risk of death from cardiovascular disease in adults with raised blood pressure, based on individual patient data from randomised controlled trials. BMJ 2001 323:75-81.

DIAGNOSTIC TOOL FOR ERECTILE FUNCTION

What constitutes erectile dysfunction of severity sufficient for treatment? In the randomised trials on newer treatments, erectile dysfunction suitable for treatment is often a diagnosis of exclusion. Various trial exclusions tell us which patients have been treated, and erectile function scoring systems tell us how well they did (or at least how much averaged scores moved, which is not always helpful).

In *Bandolier* 53 the international index of erectile function (IIEF) was described because it was an outcome of the sildenafil trials. The original IIEF had 15 questions, and was useful for clinical trials. Those 15 questions have now been examined for their usefulness as a simple patient-administered diagnostic tool of erectile dysfunction, using information gathered in randomised trials [1].

Study

In trials men had to be 18 or older, in a stable heterosexual relationship for at least six months and have a clinical diagnosis of erectile dysfunction. Erectile dysfunction was of organic, psychogenic or mixed aetiology, but anatomic disorders or patients with severe concomitant disease were excluded. A control group of men without a history of erectile dysfunction was recruited. There were 932 men with erectile dysfunction and 115 controls. Using baseline data from the randomised trials, items on the IIEF scale were examined for their ability to discriminate between men with and without erectile dysfunction.

Results

Of the 15 questions, six had moderate or good discrimination between men with and without erectile dysfunction, while for nine it was very poor to the extent of being non-existent. The ability to maintain erections during sexual intercourse was the best discriminator (100%).

Five questions were chosen (Table 1) in which the maximum score was 25 and the minimum 5. Men without erectile dysfunction had a mean score of 23 and men with erectile dysfunction had a mean score of 11. These were evaluated in a number of ways, but principally to define a cut point above which erectile dysfunction would be unlikely, and below which it would be likely.

That cut point was determined to be a score of 21. This score had a sensitivity of 98% and specificity of 88%, giving a likelihood ratio for a positive test of 8 and for a negative result of 0.02.

Let us assume that there is a 50% chance of men visiting their GP about erectile dysfunction truly suffering from it. If such a man scored 21 or less, then their chance of truly having erectile dysfunction rises to about 93%. If they score 22 or more, then it falls to 2% or less.

Comment

This is useful stuff.

What is terrific is that at least one pharmaceutical company has done the right thing and made information available so that diagnostic algorithms may be developed. There are precious few examples, and we'd like to hear of more. The information could well be useful in a primary care setting when deciding on men who may be treated in primary care, or who may need referral.

What is disappointing is that this form of study is open to bias (*Bandolier* 70), because it compared men with erectile dysfunction to men without it. We know that this architecture can lead to over-estimation of diagnostic accuracy. What is needed is a trial, like those done by CARE (*Bandolier* 70).

References:
1   RC Rosen et al. Development and evaluation of an abridged, 5-item version of the international index of erectile function (IIEF-5) as a diagnostic tool for erectile dysfunction. International Journal of Impotence Research 1999 11: 319-326.

Table 1: IIEF-5 scoring system

Over the past six months:	Score				
	1	2	3	4	5
How do you rate your confidence that you could get and keep an erection?	Very low	Low	Moderate	High	Very high
When you had erections with sexual stimulation, how often were your erections hard enough for penetration?	Almost never or never	Much less than half the time	About half the time	Much more than half the time	Almost always or always
During sexual intercourse, how often were you able to maintain your erection after you had penetrated (entered) your partner?	Almost never or never	Much less than half the time	About half the time	Much more than half the time	Almost always or always
During sexual intercourse how difficult was it to maintain your erection to the completion of intercourse?	Extremely difficult	Very difficult	Difficult	Slightly difficult	Not difficult
When you attempted sexual intercourse, how often was it satisfactory for you?	Almost never or never	Much less than half the time	About half the time	Much more than half the time	Almost always or always

The IIEF-5 score is the sum of questions 1 to 5. The lowest score is 5 and the highest score 25.

WHEN TO MEASURE BONE DENSITY

Women with low bone density have an increased propensity for fractures when older. Knowing that bone density is low is helpful in decisions about treatment. But in which women do we measure it? There are a number of clinical decision rules, but which is best? Testing them in a population with known bone density tells us that some are better than others [1].

Study

An age and sex stratified sample of Canadian women who were aged 25 years or older were selected randomly in 1996 and 1997. For the study of bone density menopausal women aged 45 years or older were eligible. Women taking HRT for less than five years were excluded, but those taking HRT for more than five years were included, and there were sensible reasons for this.

Bone mineral density was measured at the femoral neck using dual-energy X-ray absorptiometry. The results were divided between those with normal bone mineral density

Table 1: Clinical decision systems tested

Factor	Score
National Osteoporosis Foundation (NOF), test if score ≥1	
Age ≥65 years	1
Weight <57.6 kg	1
History of fracture	1
Family history of fracture	1
Current cigarette smoker	1
Simple Calculated Osteoporosis Risk Estimation (SCORE), test if score≥6	
Race not black	5
Rheumatoid arthritis present	4
History of fracture at wrist, hip of rib	4 each
Age ≥65 years	3 times first digit of age
Oestrogen therapy never used	1
Weight	(-1 times weight in lb/10)
Osteoporosis Risk Assessment Instrument (ORAI), test if score ≥9	
Age 55-64	5
Age 65-74	10
Age 75+	15
Weight 60-70 kg	3
Weight <60 kg	9
Not currently taking oestrogen	2
Age, Body Size, No Estrogen (ABONE), test if score ≥2	
Age >65	1
Weight <63.5 kg	1
Never used OC or oestrogen therapy for at least 6 months	1
Body weight criterion, test if weight <70kg	

Figure 1: Results for women with osteoporosis and normal bone density

Figure 1: Results for women with osteoporosis and normal bone density
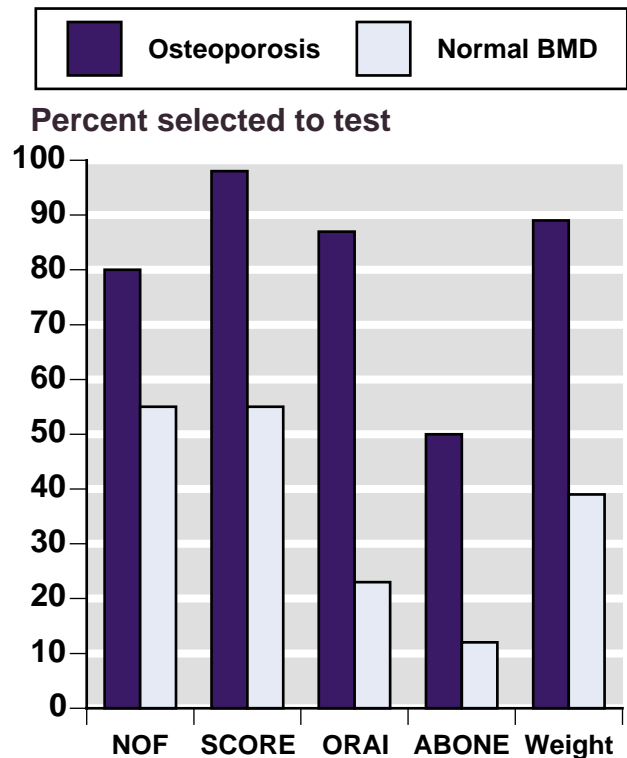
(T score less than –1 SD units), osteoporosis (T score <-2.5 SD units), and various intermediate values of moderate osteopenia (T score -2 to –2.5) and mild osteopenia (-1.0 to –2.0).

Results

There were 3,288 women, and after exclusions for those taking bone-sparing medicines, with diagnosed osteoporosis, or with missing data not allowing calculation of scores, 2,365 remained. They had a mean age of 66 and a mean weight of 69 kg; 97% were white.

Clinical decision systems tested are shown in Table 1. They came from a systematic review of the literature. SCORE and ORAI had the best discriminatory performance at each cut point. The performance of the scores at detecting women with osteoporosis and normal bone mineral density in 45-65 year old women is shown in Figure 1.

Comment

It is rare to find an examples where a single clinical decision aid has been tested in an independent sample. To find a study that systematically compares all the available decision aids is remarkable. There are several ways in which this information might be used. Many women have bone density measured whatever decision system was used, and some will argue for mass screening. This study is the essential aid to decision making.

References:
1   SM Cadarette et al. Evaluation of decision rules for referring women for bone densitometry by dual-energy X-ray absorptiometry. JAMA 2001 286: 57-63.

Book reviews

Ev*idence-based Pharmacy. Phil Wiffen. Radcliffe Medical Press, Abingdon, 2001. 182pp £19.95 ISBN 1-85775-384-4*

When it comes to what an "evidence-based" approach means to any particular professional group, general texts can sometimes be less than wholly helpful because authors write from their particular point of view. That may not always coincide with the interests of another profession. Examples may be unhelpful or unfamiliar, and concepts cast into moulds that are alien to us.

What is needed is for an author who is "one of us" to get their brains around evidence based approaches, and to rehearse them in ways that are familiar and relevant. Phil Wiffen has done this for pharmacists. As well as being involved with NHS pharmacy practice in the UK, he is also intimately involved with the Cochrane Collaboration.

That's why his book works. Much of what it contains will be familiar to those of us trying to be evidence-based. For newcomers, the way he tells the story will be a revelation. As well as the standard stuff well told, there's oodles of things a pharmacist might want to know, whether it's pharmacy journals, personal bibliographic software, essential drugs or web addresses, it's all there.

Someone who has been there, done that, and got the T-shirt takes us by the hand and keeps us safe in the jungle. It not only makes us feel and be better, but keeps us from making ludicrous mistakes because of ignorance. Given the increasing importance of pharmacists in helping decide prescribing policy, the book comes none too soon.

Risk matters in healthcare. Communicating, explaining and managing risk. Kay Mohanna & Ruth Chambers. Radcliffe Medical Press, Abingdon, 2001. 114pp ISBN 1-85775-456-5

"If anyone ever asked me for the number-needed-to-treat that would be a real indication to refer them to outpatients". That comment is from a GP discussing risk and how to communicate them, one part of an interesting book examining risks and communication and planning in a sensible and accessible way.

We all take risks. If you drive, you have about a 1 in 20,000 chance of dying in any given year, and that is a risk most of us presumably accept. The risk of dying in a train crash is about 1 in 200,000, but it gets headlines. *Bandolier* always enjoyed the health and safety talk that began with the declaration: it's the employer's responsibility to keep employees safe at work so they can go hang-gliding at the weekend.

The trouble is that risks look different, feel different, and probably are different from different perspectives. To risk is the most irregular of irregular verbs. There will probably never be any absolutely absolute and comprehensive way of describing it, but from where we are now this book makes a super stab at it.

It takes us through risks and communication, through changing the culture and managing risks in primary care. It's about how we develop our personal and organisational attitudes. Difficult, though, this, because what we are doing is to stop things that happen rarely from happening at all, and it's awfully difficult to measure that. The intellectual and organisational challenge is to understand in the first place what risk and risk management is all about.

The book may be worth having for a few pages at the end with lists of risk estimates for various medical procedures and conditions. Makes you think!

Primary care in the 21st Century

The role of nurses, doctors and pharmacists in delivering patient-centred primary care in the 21st Century. The event includes key speakers focusing on the issues of the delivery of patient-centred primary care.

Hayward Medical is organising the meeting on behalf of East Kent Health Authority who are applying for PGEA/CME approval.

Date: 21st November 2001
Venue: Kent County Cricket Club, St Lawrence Ground, Old Dover Road, Canterbury, Kent, CT1 3NZ.
Contact details: Kelli Canada, 01638 751515, kelli@haywardmedical.co.uk

Introduction to health economic evaluation

One-day course run by the Health Economics Research Centre of Oxford University.

12 October 2001 in Oxford
15 October 2001 in Southampton

The course is for anyone wanting to learn about health economics - health care decision makers; professionals in the public and private health care sectors; researchers from other disciplines. Aims of the course are to introduce key health economic concepts, and provide an appreciation of the design, conduct and analysis of economic evaluation. Places are still available for both dates.
For more information visit the HERC website (www.ihs.ox.ac.uk/herc) or email herc@ihs.ox.ac.uk